

# Patient with Purulent Pericardial Effusion Revealing Squamous Cell Lung Carcinoma

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## Abstract

Bacterial pericarditis is a rapidly progressive infection sown with heavy mortality. It usually occurs as a secondary infection to either a contiguous or hematogenous spread from a distant focus of infection. The primary involvement of the pericardium without evidence of underlying infection disease elsewhere, is sporadic. We present a unique case of a 69 -year-old patient who was diagnosed as having purulent acute bacterial pericarditis caused by Streptococcus pneumonia, revealing an underlying squamous cell lung carcinoma in the cardiology department of IBN ROCHD University hospital of Casablanca.

## INTRODUCTION

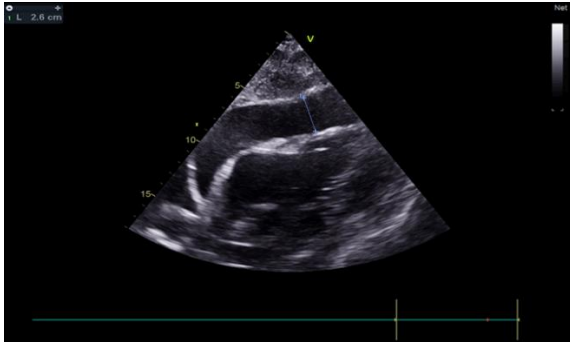
Pyopericardium is an acute infection with a rapidly progressive fulminant course and heavy mortality [1]. It is a rare subset of bacterial pericarditis and usually arises in children and or the context of an underlying disease, predisposing to immunosuppression and infection. Purulent pericarditis usually occurs as a secondary infection by contiguous spread from empyema, pneumonia, or other intrathoracic focus of infection or by hematogenous spread from a distant focus of infection [2]. The central responsibility of the pericardium without evidence of underlying infection disease elsewhere, is sporadic. We present a unique case of a 69 -year-old patient who was diagnosed as having primary purulent acute bacterial pericarditis caused by Streptococcus pneumonia revealing an underlying squamous cell lung carcinoma.

## CASE PRESENTATION

A 69-year-old man presented to our emergency service with a 2-week history of worsening of dyspnea and a sudden onset pleuritic chest pain of two days duration. The chest pain started when he was sleeping and was sharp, constant, and radiating to his back. He also complained of fever, cough productive of mucoid sputum without hemoptysis weight loss for two months and abdominal and lower limb swelling for one month. Also he had fatigue, palpitations and exertional dyspnea but denied orthopnea. He denied any history of recent

travel or exposure to sick contacts. Past medical history was significant for a recent cholecystectomy. His tobacco use is 33 pack-years and denied any alcohol or illicit drug use.

Upon physical examination, he was alert, oriented in time and place, afebrile, with a heart rate of 122 bpm, a respiratory rate of 22bpm, and blood pressure of 96/72 mmHg. Oxygen saturation on room air was 88%. An oral examination revealed multiple dental caries. A cardiopulmonary investigation revealed scattered wheezes and rhonchi that there was no cyanosis or peripheral edema. An initial laboratory workup showed elevated white blood cell (WBC) count of 17.700 cells/mm<sup>3</sup> with 93% neutrophil predominance, high inflammatory markers of erythrocyte sedimentation rate 88 mm/hour and C-reactive protein (CRP) 244 mg/dl, and moderately elevated lactate dehydrogenase. A basic metabolic panel was within regular intervals. Cardiac enzymes were healthy. Serial troponins were negative. An electrocardiogram was performed revealing sinus tachycardia, low-voltage QRS complexes, electrical alternans, and diffuse ST-segment elevations, and A chest X-Ray showed inflated bilateral lung fields, a flattened diaphragm and regular cardiac silhouette. An echocardiogram revealed a large pericardial effusion, with a heart swinging aspect and the diastolic collapse of the right ventricle (Fig 1). The inferior vena cava was dilated and non-collapsing.



**Figure 1.** Transthoracic echocardiogram showing pericardial effusion



**Figure 2.** Axial computed tomography scan of the chest showing the tumor in the right lower lobe

An emergency pericardiocentesis was performed with removal of 900 ml of purulent pericardial fluid with immediate hemodynamic improvement. The pericardial fluid was sent for culture, cell analysis, and cytology. WBC count was 15,306 cells/mm<sup>3</sup> with 98% segmented neutrophils. He was started large spectrum antibiotics with intravenous ceftriaxone and moxifloxacin. Pericardial fluid cultures grew Gram-positive cocci in chains, which were characterized as *Streptococcus Pneumoniae*, which was susceptible to penicillin and ceftriaxone. Anaerobic, fungal, and acid-alcohol resistant bacilli cultures were negative. Pericardial fluid cytology was also negative for any malignant cells. Extensive workup with urine analysis, urine culture, and blood culture to look for the source of infection failed to identify any other focus of infection. Human immunodeficiency virus (HIV), hepatitis serology, and tuberculosis adenosine deaminase testing were negative. Antibiotics were withdrawn to intravenously administered ceftriaxone (2 g every 24 hours) as per culture sensitivity.

Computed tomography showed bilateral moderate pleural effusion and a solitary round lesion in the right lower lobe of the lung (Fig 2). Bronchoscopy with bronchoscopic lung biopsy and brushing revealed a

squamous cell carcinoma. His hospital course was complicated with acute exacerbation of his chronic obstructive pulmonary disease for which he was started inhaled bronchodilating drugs (anticholinergics ipratropium bromide), and corticosteroids. However, over time, he did not have complete resolution of symptoms with the persistence of chest pain and dyspnea despite hemodynamic and echocardiographic features improvement. Following a heart team discussion, a surgical pericardial window with a subxiphoid approach was performed. He reported improvement in his symptoms after drainage. Repeat bedside transthoracic echocardiogram showed resolution of the previously noted effusion. The drain was removed after three days. He was discharged home in a stable condition and received a total of 4 weeks of intravenously administered ceftriaxone. Six months post-discharge he reported complete resolution of his cardiac symptoms and after the staging of his carcinoma, received one course of chemotherapy consisting of the combination of cisplatin 160mg and etoposide 500mg with dexamethasone and 10% mannitol. Repeat echocardiography showed a thicker pericardium with no constrictive pattern and a complete resolution of pericardial effusion.

## DISCUSSION

Acute pericarditis may be caused by a wide variety of disorders [3]. Possible aetiologies include autoimmune diseases, malignancies, cardiac trauma, uremia, and infections (including viral, bacterial, and fungal etiologies) [3]. Pericardial effusion is joint in patients with cancer, mostly in those with breast, lung, and hematologic malignancies [4]. In our case, malignant effusion was initially highest on our differential diagnoses, as malignancy is the most classical etiology of pericardial effusion and ensuing tamponade [5]. Among these patients, the process of development of pericardial effusion includes either direct cancer involvement of the pericardium, such as metastasis or indirect effects of malignancy and subsequent toxicity of chemotherapy or radiation, like pericardial fibrosis, lymphatic obstruction, and hypoalbuminemia [6]. The bacterial infection is thought to be a rare etiology of pericardial effusions in cancer patients, especially in the modern antibiotic and vaccination era, with a reported incidence of less than 1% in multiple series [2]. The most common germs incriminated are *Streptococci*, *Staphylococci*, *Haemophilus*, and *Mycobacterium tuberculosis* [7]. Pyopericardium typically occurs as a secondary infection by contiguous spread from bordering intrathoracic focus including an extension from the pulmonary, myocardial, and subdiaphragmatic site of infection or by hematogenous dissemination from a distant infection elsewhere in the body. [8] Among these, direct extension from pneumonia or pleural empyema accounts for the majority of cases. Presenting as a primary infection without evidence of underlying

disease elsewhere is very rare [7]. Immunosuppression, malignancies, alcoholism, uremia, chest trauma, and cardiothoracic surgery are some of the usual predisposing conditions for bacterial pericardial effusion [9].

Pyopericardium is an acute infection characterised by the presence of frank pus in the pericardial cavity. It has a fulminant course and is associated with somber prognosis with a large number of cases only discovered postmortem. If not diagnosed quickly and treated promptly the mortality rates can approach 100%. The complication and mortality rate is still very high with proper treatment, with the mortality rate approaching 40%. Cardiac tamponade or septic shock is the most common cause of death in these patients [10]. Our case is a scarce combination of a patient with primary lung cancer who presents with nonmalignant, pyogenic pericarditis caused by *S. pneumonia*, which is a common pathogen in patients with pneumonia, meningitis, sinusitis and otitis. However, it is a rare infectious source in patients with pericarditis. From a search of the medical literature, we identified a total of 24 cases of bacterial pericarditis caused by *Streptococcus* group from 1994 to 2019. Among these, five cases were attributed to *S. pneumoniae*. [7, 9, 11-13]

To the best of our knowledge, this is the first published case of pyopericardium caused by *S. pneumonia*, revealing an underlying squamous cell carcinoma of the lung. We believe that the most likely etiology of infection in our case was transient bacteremia from a pleura-pulmonary breach in our patient's tumor with hematogenous spread and seeding of bacteria in pericardial cavity leading to suppuration [14-24].

## CONCLUSIONS

Acute suppurative bacterial pericarditis, although rare, should always be kept in mind as a possible cause of pericarditis. As this disease has a rapidly progressive fulminant course, a high index of clinical suspicion along with prompt intervention is critical for a successful outcome.

## Conflict of Interest

There is no conflict of interests with this article

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